

Note to Readers: If you need assistance accessing items in this Supplemental Material, please contact ehp508@niehs.nih.gov. Our staff will work with you to assess and meet your accessibility needs within 3 working days.

Table of Contents for Supplemental Material

Prenatal and Postnatal Exposure to Persistent Organic Pollutants and Infant Growth: A Pooled Analysis of Seven European Birth Cohorts

Nina Iszatt, Hein Stigum, Marc-André Verner, Richard A. White, Eva Govarts, Lubica Palkovicova Murinova, Greet Schoeters, Tomas Trnovec, Juliette Legler, Fabienne Pelé, Jérémie Botton, Cécile Chevrier, Jürgen Wittsiepe, Ulrich Ranft, Stéphanie Vandentorren, Monika Kasper-Sonnenberg, Claudia Klümper, Nynke Weisglas-Kuperus, Anuschka Polder,¹⁷ Merete Eggesbø, and OBELIX

Table S1. Description of the birth cohorts with biological PCB-153 and p,p'-DDE exposure biomarkers included in the present study.*

Table S2. Chemical-analytical methods and detection/quantification limits of the birth cohorts.*

Figure S1. A) Conceptual representation of the pharmacokinetic model and B) examples of blood POP levels in mothers and infants.*

*Adapted from Verner et al. 2013. Reproduced with permission from Environmental Health Perspectives. AUC area under the curve. Simulations were carried out with a maternal daily dose of 10 ng/kg body weight/day. Model assumptions: exclusive maternal exposure through diet; complete gastrointestinal absorption; exclusive and homogenous distribution of POPs in maternal and child lipids with unlimited transplacental diffusion (due to lipophilicity). POPs elimination (e.g., fecal excretion, metabolism) was based on published half-life values. Breast milk consumption rate was based on exclusive/partial breastfeeding data from the general population (Arcus-Arth et al. 2005).

Table S3. Description of heterogeneity between cohorts modelled with random slope (deviation from fixed effect).

Table S4. Comparison of pooled results from models fitted with random intercepts (Model 1) or additionally random slope for PCB-153 and *p,p'*-DDE (Model 2).

Figure S2A. Directed acyclic graph of the association between infant growth and total exposure.

Figure S2B. Directed acyclic graph of the association between infant growth and prenatal exposure.*

*Birth weight and gestational age are intermediate variables between prenatal POP exposure and infant growth. We are interested in the effect of exposures on infants' postnatal growth, not one that may be merely a continuation of prenatal growth mediated via birth weight. We included these variables in the model to close the pathway from prenatal exposure to infant growth via birth weight so that the model estimates only the direct association between prenatal exposure and postnatal growth.

Figure S2C. Directed acyclic graph of the association between infant growth and postnatal exposure.

Table S5. Biomarker concentrations and cord blood POPs concentrations estimated in the pharmacokinetic model (ng/g lipid).

Table S6. Pearson's correlation coefficients between prenatal concentrations, postnatal exposure and total breastfeeding duration.

Table S7. Variance inflation factors (VIFs) for prenatal and postnatal exposure in the same model.

Table S8. Comparison of results: leaving out one cohort at a time for PCB-153 and *p,p'*-DDE.

Table S9. Comparison of results for PCB-153 and *p,p'*-DDE: complete case dataset, multiple imputation dataset, and biomarker (instead of modelled) concentrations as "prenatal exposure."

References